Treatment of Mushroom Amanitin Poisoning

TO THE EDITOR: We have three comments on the excellent paper by Pond and co-workers on mushroom amanitin poisoning in the August issue.[1]

Biliary recirculation of amanitin toxins was treated by duodenal tube and by activated charcoal. In a November 1983 poisoning—not reported in the series—Dr Larimore Cummins, a Santa Cruz, California, gastroenterologist, cannulated the common bile duct of a patient who survived *Amanita phalloides* poisoning. Where available, this procedure should be considered in severe poisonings as bile-laden amatoxin is totally diverted.

One mushroom cap from the collection eaten by patients 9 through 22 was in fact available at Brookside Hospital (San Pablo, California) and was identified as *Amanita phalloides* by one of us (P.V.), who also found strongly positive findings on a Meixner test.

Finally, there is now one paper available[2] on 205 consecutive European poisonings, where the use of intravenously given penicillin-G sodium resulted in a statistically significant decline in mortality. This study by Floersheim and coworkers is the largest series to date, and carefully analyzed by multiple regression. Previously, sufficient numbers of patients to statistically evaluate have been lacking in all published series. Thioctic acid appeared to be associated with an excess mortality, but one cannot rule out adverse selection. The intravenous administration of 40 million units a day of penicillin, therefore, is an option that may be considered.

THOMAS J. DUFFY, MD
PAUL VERGEER
Toxicology Committee
Mycological Society of San Francisco
5706 Carlos Ave
Richmond, CA 94804

REFERENCES

1. Pond SM, Olson KR, Woo OF, et al: Amatoxin poisoning in northern California, 1982-1983. West J Med 1986 Aug; 145:204-209

2. Floersheim GL, Weber O, Tschumi P, et al: Die klinische Knollenblaetterpilzvergiftung (Amanita phalloides)—Prognostische Faktoren und therapeutische Massnahmen. Schweiz Med Wochenschr 1982; 112:1164-1177

Manipulative Therapy for Low Back Pain

TO THE EDITOR: It would seem that the sheer magnitude of the suffering and cost of low back pain to our society would mandate that allopathic and osteopathic communities would finally unite on the issue of manipulative therapy.

To that end, let us share some observations that may apply and that we hope may encourage further dialogue.

Fifteen osteopathic medical schools are teaching subjective and objective methods for diagnosing spinal malfunctions leading to pain and disability. Additionally, their students are being taught the osteopathic principles involved in specific treatment. From there, it is hoped that they are trained in the skills to treat "hands on" for the dysfunction.

Manipulative therapy, being noninvasive, has a low morbidity risk. Outcomes, though occasionally dramatic, usually are consistent with "dosage" and patient cooperation.[1] As in any treatment modality, specificity of diagnosis contributes greatly to effectiveness. Recent manipulative research protocols[2] have standardized treatment, and outcomes are consistent and largely predictable. Postgraduate courses in osteopathic principles and practice are available at virtually all osteopathic medical schools for physicians interested in "hands on" experience.

Manipulative therapy adds another modality to the armamentarium of physicians using drugs, surgical treatment and other physical modalities—quite frequently a highly effective modality, indeed.

ARTHUR G. MADORSKY, DO, MD
Professor of Internal Medicine
LORANE M. DICK, DO
Chair, Department of Osteopathic Principles and Practice
College of Osteopathic Medicine of the Pacific
College Plaza
Pomona, CA 91766-1889

REFERENCES

1. Kimberly PE: Formulating a prescription for osteopathic manipulative treatment. J Am Osteopath Assoc 1980; 79:506-513

2. Branson MH, Buerger AA: Randomized clinical trials in the validation of cervical and lumbar manipulation. *In* Greenman PE (Ed): Concepts and Mechanisms of Neuromuscular Functions. New York, Springer-Verlag, 1985, pp 90-105

Applying Information Technology to Clinical Medicine

TO THE EDITOR: The fascinating case of glucagonoma reported by Bolt and co-workers in the June 1986 issue[1] presents the tragic story of a man tormented by symptoms for more than a decade until an astute physician equated "sore tongue," "chronic indigestion" and "variable, red rash" with the possibility of an islet cell tumor. The patient underwent extensive evaluation at a university center documenting the presence of a glucagonoma. Ironically, the man subsequently died of complications of treatment.

The authors point out that the 12-year delay in diagnosis is typical of illness due to islet cell tumors. Surprisingly, they blame "the recent emphasis on cost effectiveness" as a "factor inhibiting early diagnosis."

The patient had been seen by numerous physicians, including appropriate subspecialists, for more than 12 years before anyone knew what was wrong with him. I seriously doubt that glucagon levels were not obtained for reasons of cost containment. More likely, the physicians did not think seriously about the possibility of glucagonoma. Or, if they did, they erroneously discounted the idea because they were unaware that more than 10% of patients with these tumors do not present "classic" features described in the medical literature.

The cost of this poor man's illness included 12 years of unmitigated morbidity, plus the cost of numerous useless investigations including multiple glucose tolerance tests, multiple skin biopsies, radiographic evaluation of the entire gastrointestinal tract and gallbladder, endoscopic evaluation of the stomach, small bowel and sigmoid colon, abdominal ultrasonograms and routine blood tests. Even after the diagnosis had been suspected and chemically confirmed by a glucagon level of 2,400 pg per ml, the man underwent additional tests including computer-assisted tomography of the head and abdomen with and without contrast; measurement of serum levels of amino acids, gastrin, insulin, vasoactive intestinal peptide, pancreatic polypeptide, cortisol, thyroxine, free thy-